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ONLINE ARTICLES

Axillary artery intimal dissection with thrombosis and brachial plexus injury after reverse total shoulder arthroplasty

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The most commonly reported complications of reverse total shoulder arthroplasty (RTSA) are scapular notching, glenoid component loosening, heterotopic ossification, hematoma, infection, and instability.[5,13,14] Less commonly seen complications include iatrogenic acromial fractures, complications of the humeral component, and neurologic complications.[2,13,14] Although axillary artery thrombosis has been well documented as a complication of proximal humeral fractures and glenohumeral dislocations, it is not a commonly reported complication of RTSA. The anatomic location of the axillary artery does place it at risk of injury during trauma by any means to the shoulder. Review of the literature has identified 1 reported case of late axillary artery thrombosis approximately 1 month after RTSA.[7] We report on 1 case of axillary artery thrombosis and brachial plexus injury in the immediate postoperative period after RTSA.

Case report

A 65-year-old woman with a medical history of obesity (body mass index of 41) and hypertension who did not smoke underwent right RTSA for rotator cuff arthropathy. A 15-cm incision was used for the deltopectoral approach to the right shoulder because of her body habitus, but the exposure was otherwise routine. The pectoralis major tendon was slightly tighter than often seen, and the upper 2 cm was recessed. The humeral head cut was made in the typical fashion with 30° of retroversion and at 45° to the long axis of the humeral shaft. Osteophytes about the humeral head cut were minimal and easily removed. Glenoid exposure was easily achieved with the arm on a padded Mayo stand, and light reaming was performed before placement of the baseplate with 4 parallel locking screws: a 22-mm screw at the 12-o'clock position, 14-mm screws at the 3- and 9-o'clock positions, and a 30-mm screw at the 6-o'clock position. A 32-4 glenosphere was attached, and a locking screw was placed in standard fashion. The shoulder was then externally rotated for humeral reaming and trialing. A size 10 stem with a standard liner was found to reduce easily with good stability and appropriate tension on the coracobrachialis. The humeral implant was inserted to the correct height in 30° of retroversion. A standard 32 liner was inserted, and the shoulder was then easily reduced. After placement of all implants, 150° of forward elevation and 50° of internal rotation were achieved. The subscapularis was repaired and allowed 60° of external rotation in adduction and 85° of external rotation in abduction. The wound was closed in multiple layers, and local analgesia with 50 mL of bupivicaine hydrochloride 0.25% with epinephrine was injected in the subcutaneous tissues.

No institutional review board approval was required for this case report.

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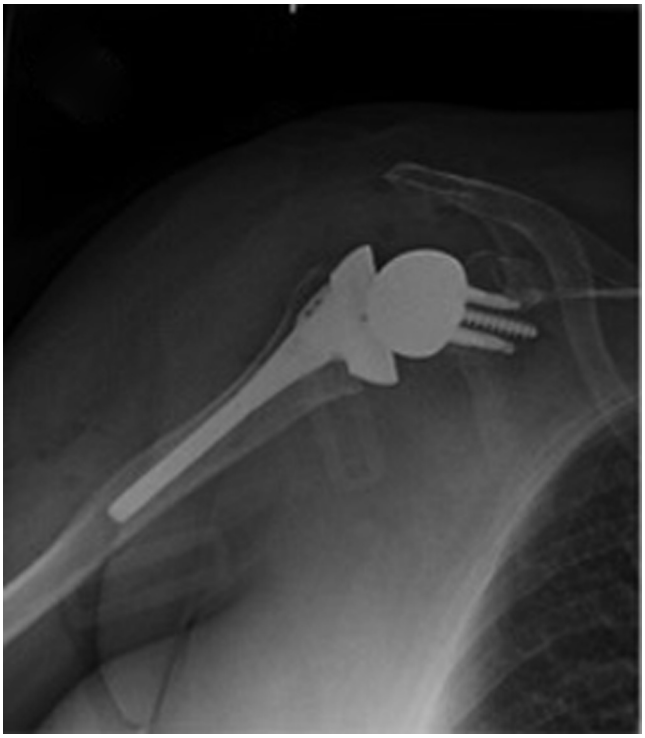

Figure 1 Immediate postoperative anteroposterior radiograph of the right shoulder.

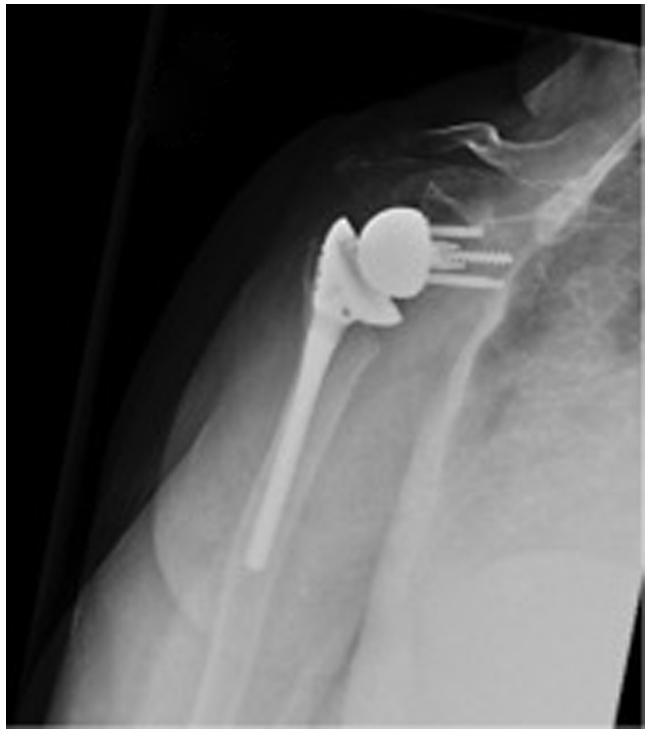

Figure 2 Right shoulder anteroposterior radiograph following revision procedure after shortening of humerus.

In the postoperative care area, the patient noted diminished sensation in a stocking-glove distribution from the elbow distally. She complained of a cold sensation to her wrist and hand but on examination had palpable radial and ulnar artery pulses at the wrist. The right hand and forearm were elevated on pillows at heart level, and the patient was serially monitored. Approximately 1 hour later, she had absent brachial, radial, and ulnar pulses that were not appreciable on palpation nor were Doppler signals found. The patient had decreased motor function in her right hand, with an inability to extend the wrist, extend the thumb, or flex or extend the digits. At that time, the differential diagnosis based on her clinical examination findings was axillary artery thrombosis and brachial plexus injury, possibly due to excessive humeral lengthening (Fig. 1). The decision was made to return the patient to the operating room to undergo vascular surgery for formal exploration of the axillary and brachial arteries to look for possible thrombosis and shortening of the humeral implant to decrease any stretch on the neurovascular structures.

In the second procedure, the prior deltopectoral incision was reopened and the shoulder dislocated. This did not improve the vascular examination. The humeral implant was tamped out of the medullary canal and the proximal humerus shortened approximately 1 cm. The implant was impacted back into the humerus and the shoulder reduced (Fig. 2).

The brachial artery was then explored through a longitudinal incision over the proximal aspect of the medial arm. The distal right axillary and proximal brachial arteries were thrombosed with a long segment of intimal dissection. The patient underwent systemic heparinization. A transverse arteriotomy was performed on the brachial artery, and thrombectomy was performed. A large intimal flap and thrombus were retrieved (Fig. 3), after which excellent inflow was noted. The artery was repaired primarily, and completion duplex ultrasound showed patent radial and ulnar arteries. Doppler ultrasonography showed satisfactory signals at the radial and ulnar arteries as well. The patient had no evidence of compartment syndrome and soft compartments, so fasciotomy was deferred at that time. Subscapularis repair and skin closure were routine. Heparin was not continued postoperatively.

By the following morning, tense hematomas had developed at both surgical sites, so the patient was taken to the operating theater a third time for irrigation and débridement of both incisions. No significant bleeding sources were identified, but Arista Absorbable Hemostat (Davol, Warwick, RI, USA) and Floseal (Baxter Healthcare, Hayward, CA, USA) were applied to minimize oozing at both sites. The compartments remained soft and were closely monitored. Since that time, good arterial inflow appreciated by palpable pulses at the wrist was maintained, but the patient had persistently abnormal sensorimotor function of the right hand, with apparent radial and ulnar nerve palsies and decreased sensation of the hand and forearm with preserved medial nerve function. The 1-month follow-up computed tomography angiogram showed an intact repair without evidence of aneurysmal

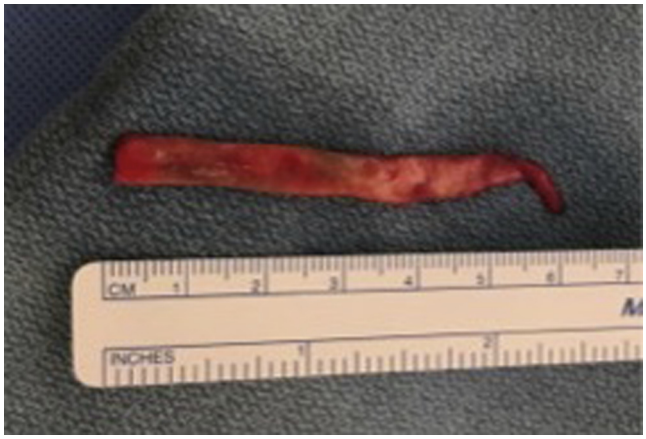

Figure 3 Intimal flap and thrombus removed from distal right axillary and proximal brachial arteries.

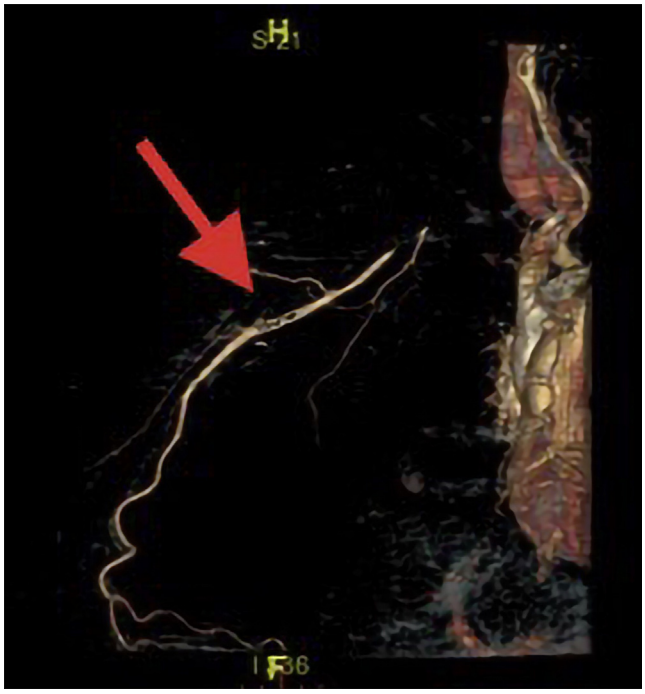

Figure 4 One-month follow-up computed tomography angiogram demonstrating intact repair (*arrow*), without evidence of aneurysmal degeneration of distal axillary artery.

degeneration of the distal axillary artery (Fig. 4). The radial nerve and ulnar nerve function had partially returned at 3 months and was normal at the 6-month mark.

Discussion

This case demonstrates a rare and devastating complication of an axillary artery intimal tear with dissection and thrombosis, as well as brachial plexus neurapraxia, following RTSA. Recent studies have demonstrated increased use of RTSA with expanding indications owing to improved implants and increased surgeon comfort with the procedure, as well as many successful outcomes regarding function, pain relief, and range of motion.[3] Despite the increase in use, the fact remains that reported complication rates after RTSA range from 19% to 68%; complications include scapular notching, instability, infection, hematoma, mechanical baseplate failure, acromial stress fracture, periprosthetic fracture, and neurovascular injury.[4]

The axillary artery and brachial plexus are susceptible to injury during shoulder surgery because of their close proximity to the glenoid, coracoid, and clavicle. They travel in a relatively restricted space between the clavicle and chest wall and are tethered proximally by both the prevertebral fascia and the nerve roots, as well as distally by the axillary sheath.[13] RTSA can cause humeral lengthening, resulting in increased tension on these neurovascular structures and potential traction injury during the procedure.

Neurovascular injury to the axillary artery and brachial plexus has been well described following trauma to the shoulder, with the most common mechanism being anterior shoulder dislocation. Various mechanisms have been proposed to explain this. It is thought that the pectoralis minor could act as a fulcrum over which the axillary artery could be bent or compressed by the humeral head.[8] Furthermore, it has been proposed that the axillary artery, being tethered by the subscapular and humeral circumflex arteries, can be elongated as it is pulled with humeral retraction or rotation[1] (Fig. 2). Moreover, it has been suggested that the axillary artery may be more susceptible to injury from shoulder trauma or surgery in patients older than 50 years owing to atherosclerosis and loss of arterial elasticity.[9] Blunt trauma to the shoulder has also been described as a mechanism for axillary artery injury. Gallucci et al[6] described a delayed axillary artery thrombosis 2 months after a nondisplaced humeral neck fracture. They also postulated that the initial trauma led to intimal damage but noted that it often takes an extended period for a subintimal dissection to occlude an artery and cause ischemia.

Specifically, vascular complications after shoulder arthroplasty are rare and seem to most commonly be characterized as hematoma or phlebitis. Wingert et al[12] described a case of an intraoperative avulsion of the axillary artery during RTSA that they attributed to traction injury to the artery during the procedure. In addition, Ghanem et al[7] reported a case of a delayed axillary artery occlusion after RTSA that gradually progressed in the postoperative period and was diagnosed 10 weeks postoperatively via angiography. They hypothesized that the thrombosis was likely due to an intimal injury during the procedure. Bents[2] reported 2 cases of acute axillary artery thrombosis after humeral resurfacing arthroplasty in patients aged 59 and 64 years. The author postulated that the position of the humerus during the procedure re-creates forces on the axillary artery similar to those seen with an anterior shoulder dislocation.

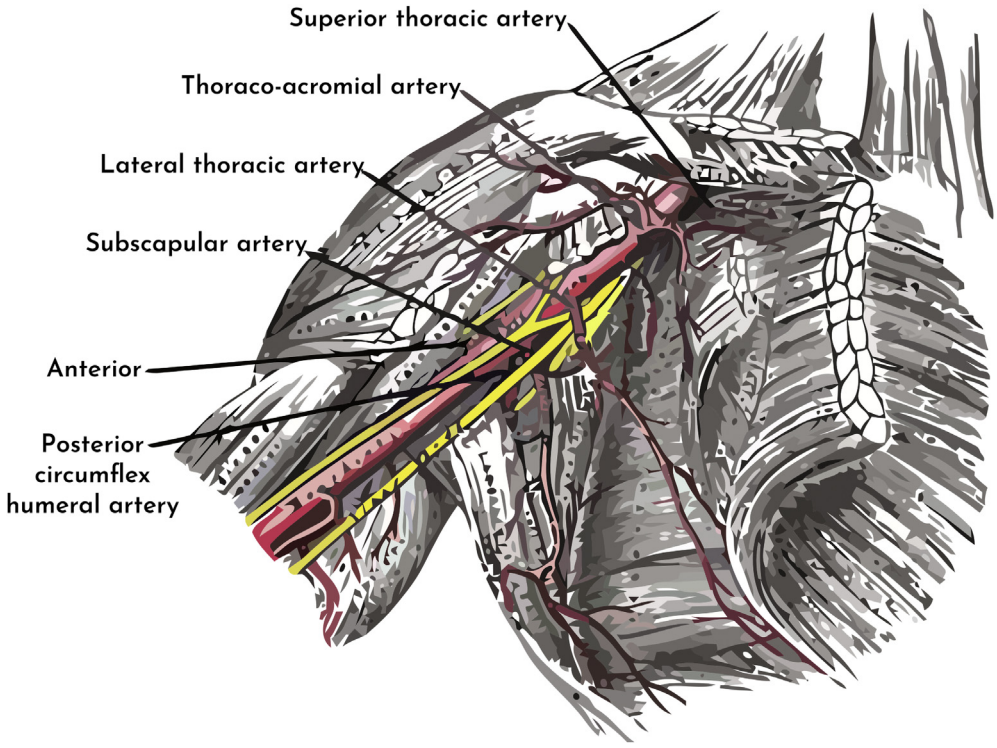

Figure 5 Branches of axillary artery. Illustration by Arien Cherones.

In this case, we did not believe that the patient had untoward humeral lengthening at the initial operation (Fig. 4), but we shortened the humerus during revision to decrease any tension from this mechanism (Fig. 5). The etiology of our patient's brachial plexus injury remains unclear, but her anatomy likely did not have much tolerance for standard positioning and our normal use of retractors. We hypothesize that her neurologic symptoms could potentially have been caused by traction injury and doubt that they were ischemic in nature because of the concurrent axillary artery dissection and thrombosis. Furthermore, we believe that the axillary artery dissection and thrombosis likely occurred from either traction or an intimal injury during the procedure by one of the mechanisms previously described.

Postoperative hematomas following systemic heparinization infusions during vascular surgery were not completely unexpected and possibly could have been mitigated with heparin reversal—though at the expense of possible arterial thrombosis in the freshly dissected artery. Placement of drains may have decreased the need for reoperation but may have led to considerable blood loss and transfusions.

The diagnosis of acute limb ischemia due to arterial thrombosis is primarily clinical and can be confirmed by arterial duplex examination at the bedside.[11] Computed tomography angiography is a useful adjunct to confirm the diagnosis and delineate the anatomy in circumstances in which the mechanism of acute limb ischemia cannot be delineated; however, this should not delay treatment. The treatment in this case was emergent because of the motor and sensory deficits associated with the arterial thrombosis. Less invasive means such as thrombolysis would not have been successful in this case because of the intimal injury and the delay associated with the thrombolysis. Vascular stent application in this area is discouraged because of the high mobility in this area; however, in some circumstances, this may be an option when open repair cannot be performed.[10] Ultimately, a successful outcome depends on a meticulous physical examination, expeditious diagnosis, and prompt treatment.

Conclusion

Neurovascular injury following RTSA is uncommon but potentially devastating. This case highlights the importance of routine neurovascular examination in the immediate postoperative period. A high index of suspicion is paramount to obtaining an early diagnosis, minimizing morbidity, and maximizing the chances of successful treatment.

Disclaimer

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